

Acute Recurrent Pericarditis Accompanied by Graves' Disease

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The etiology of acute pericarditis is often thought to be autoimmune, and Graves' disease has been reported in a few series to manifest as acute pericarditis. Since the etiology of recurrent pericarditis is known to be more associated with autoimmune causes, recurrent acute pericarditis may be a potential cardiovascular complication of Graves' disease. We report a case of recurrent acute pericarditis that was presumed to be associated with Graves' disease which was controlled after management of the problem of the thyroid. (**Korean Circ J 2012;42:419-422**)

KEY WORDS: Pericarditis; Graves disease.

Introduction

Acute pericarditis is the most common problem among diseases related to the pericardium. Its common causes are viral, bacterial, tuberculosis, myocardial infarction, malignancy, trauma, and autoimmune causes.¹⁾ Graves' disease is an autoimmune thyroid disease which causes overproduction of the thyroid hormone. Well-known cardiovascular complications of Graves' disease are ventricular arrhythmia, tachycardia-associated cardiomyopathy, and mitral valve prolapse.²⁾ As the etiology of Graves' disease is known to be autoimmune, autoimmune pericarditis may be a potential cardiovascular complication of the disease. A few reports have addressed the possibility of acute pericarditis associated with Graves' disease^{2,3)} but there are no reports of associated recurrent pericarditis. We report a case of acute recurrent pericarditis that was presumed to be associated with Graves' disease and was controlled after management of the disease.

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Case

A 42-year-old male visited the emergency department complaining of chest pain. He had previously been admitted 3 months ago due to similar symptoms, and was diagnosed with acute pericarditis according to the features of typical chest pain and findings of electrocardiography (ECG) and echocardiography (Fig. 1A). Ibuprofen and colchicine were administered and the symptoms improved within 24 hours. At the time, he was discharged after 5 days, and he had stopped taking the medications by himself after 2 weeks as he did not feel any continuing symptoms.

At admission, he complained of severe chest pain which had begun 4 hours prior to admission. The pain increased with deep breathing and position change. He also complained of palpitation which had been present prior to the development of chest pain. The patient had no relevant abnormal medical history and no current medication, and reported to have lost 2 kg during the past 2 months. He was a 14-pack-year smoker, but denied any alcohol use.

Upon general examination, he appeared acutely ill. His vital signs in the emergency department were as follows: body temperature 36.9°C, pulse rate of 121 beats per minute, and blood pressure 140/89 mm Hg. The heartbeat was regular and no cardiac murmur including precordial friction rub could be identified. In addition, the lung sound was clear. There was no thyroid enlargement or neck vein engorgement.

Laboratory results including complete blood count, biochemistry profile, and cardiac enzymes were within normal limit. The chest radiograph finding was normal. ECG showed sinus tachycardia and diffuse concave ST-segment elevation in all leads except aVR and V1.

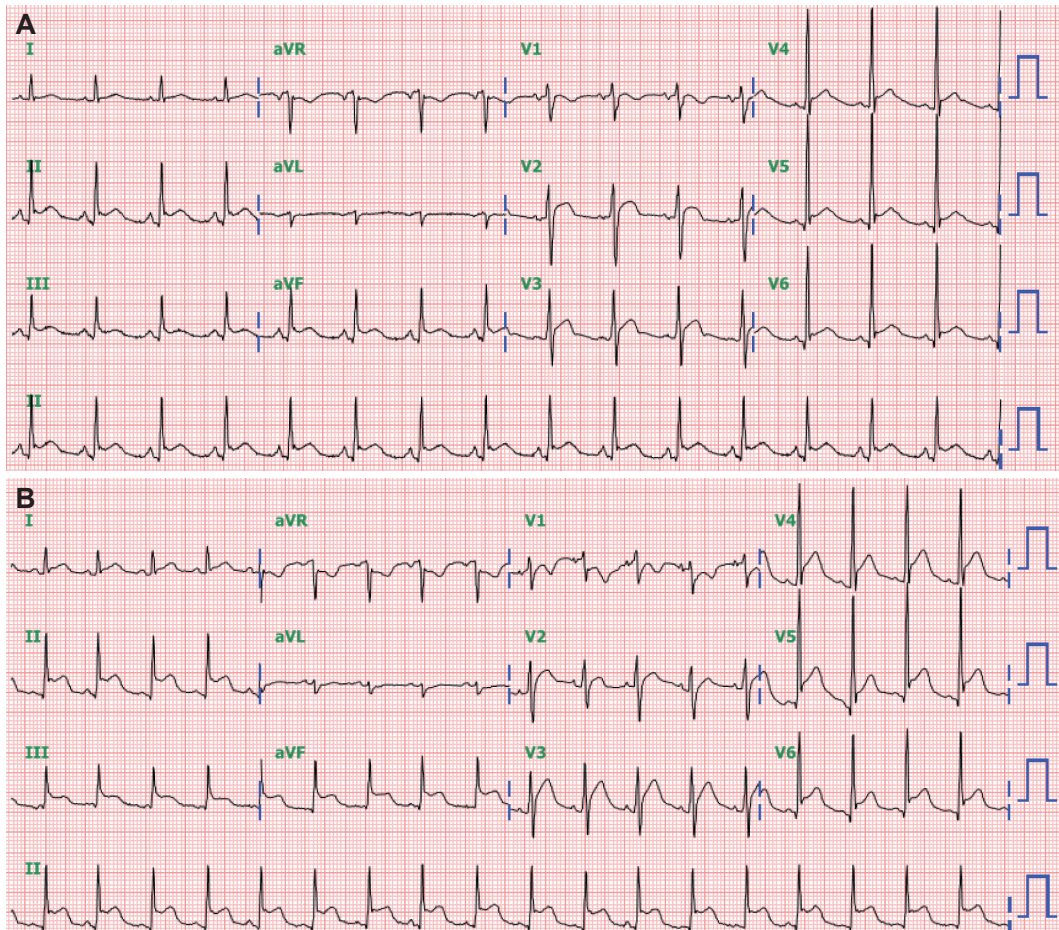


Fig. 1. Twelve-lead electrocardiograms (ECG) in patient presenting with acute recurrent pericarditis. A: ECG at the 1st admission showed diffuse ST-segment elevation. B: ECG at the 2nd admission also showed diffuse ST-segment elevation except aVR and V1.

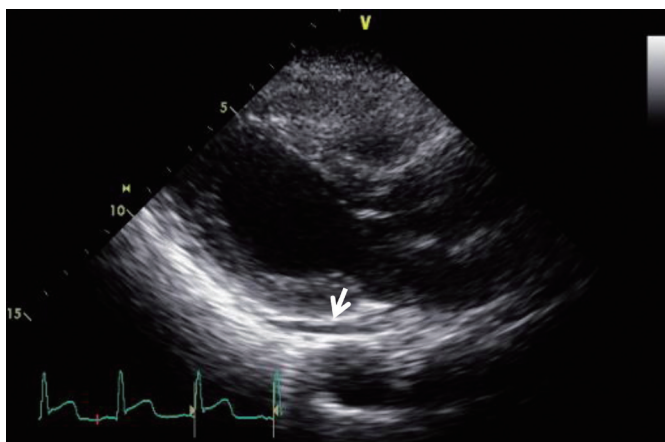


Fig. 2. Initial two-dimensional transthoracic echocardiogram in the parasternal view showed a small pericardial effusion (arrow).

PR segment was normal (Fig. 1B). Transthoracic echocardiography showed normal systolic and diastolic function of the left ventricle (LV) and a scanty amount of pericardial effusion at the LV posterior side (Fig. 2). Echocardiography that had been performed at the previous admission had shown constrictive physiology, but no such

finding was visible this time. Cardiac magnetic resonance imaging showed a slightly thickened pericardium with small amounts of pericardial effusion (Fig. 3). Autoimmune antibodies (ANA and Anti-ds-DNA) showed normal results. According to the characteristics of the chest pain, which was sharp and pleuritic in nature, and the diffuse concave ST elevation without relevance to any certain coronary artery which was out of proportion to the pain intensity, the patient was diagnosed as acute pericarditis rather than coronary spasm-related angina.

The patient was given ibuprofen and colchicine. Due to a history of palpitation and weight loss, tests for thyroid function were done on hospital day 3. Serum thyroid hormone examination showed evidence of hyperthyroidism with a T4 level of 14.2 $\mu\text{g/mL}$, T3 level of 182 ng/dL, and TSH level of 0.06 $\mu\text{IU/mL}$ (normal value T4 4.7-12.5 $\mu\text{g/mL}$, T3 76-190 ng/dL, TSH 0.3-6 $\mu\text{IU/mL}$). Thyroid autoantibody evaluation and a thyroid scan were performed. TSH receptor antibody was 5.4 IU/L (normal 0-1 IU/L), thyroglobulin antibody 89 U/mL (normal 0-60 U/mL), and microsomal antibody 136 U/mL (normal 0-60 U/mL) all showed elevated levels. Thyroid scan showed 17% (normal 5 to 15%) uptake and the patient was diagnosed with

Graves' disease, after which methimazole 10 mg bid was prescribed. Chest pain subsided within 24 hours, and the patient showed marked clinical improvement within 7 days with improvement of palpitation and general weakness. Elevated ST-segment was improved. The patient was discharged with medications for anti-hyperthyroidism and colchicine. No clinical evidence of recurrence was found during the following 3 months. The ST-segment eventually normalized on the follow up electrocardiogram (Fig. 4).

Discussion

Acute pericarditis can develop from a wide variety of inflamma-

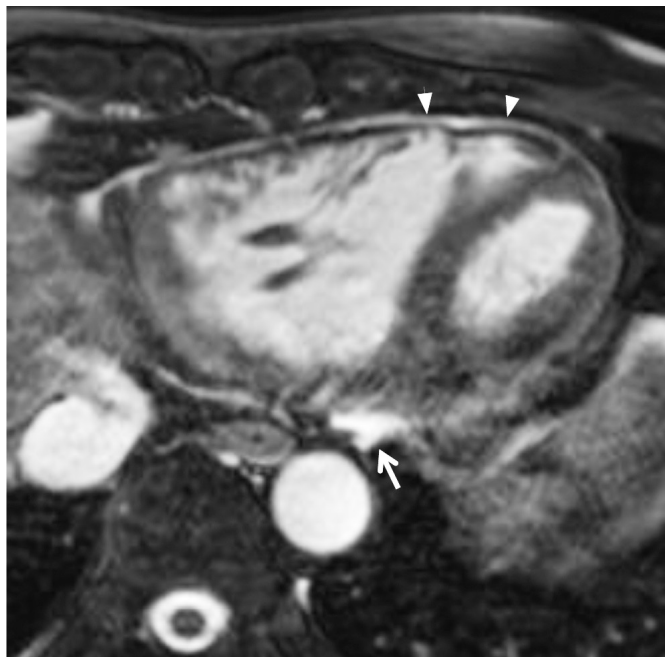


Fig. 3. Whole heart coronary magnetic resonance angiographic imaging demonstrates a slightly thickened pericardium (arrowheads) and minimal amount pericardial effusion (arrow).

tory diseases. The etiology of acute pericarditis is not clear in many of the cases, with less than 20% of patients designated a specific etiological diagnosis.⁴⁾ It can also present as the first manifestation of a systemic disease such as SLE and other autoimmune diseases. Acute pericarditis has been reported to recur in 30%, and in 15-30% of subjects with pericarditis of unknown origin not treated with colchicine.⁵⁾ Recurrent pericarditis, with its incubation period of several months, the presence of autoantibodies in patients subjected to the disease, and its response to steroids, has been reported to have a closer relationship with autoimmune diseases.¹⁾

Graves' disease is a well-known autoimmune thyroid disease, and its complications such as ophthalmopathy and myxedema are attributed to lymphocyte infiltration. Graves' disease is also associated with various complications of the heart, and there have been few reports of acute pericarditis associated with Graves' disease. Clarke et al.³⁾ reported 4 cases of acute pericarditis associated with Graves' disease and postulated a similar pathogenetic background of the inflammation with other complications of Graves' disease. Tsai et al.²⁾ also reported a patient that was not treated for Graves' disease and who presented with acute pericarditis.

In our report, we present a case of acute recurrent pericarditis that was considered to be associated with Graves' disease. Thyroid function was not checked during the first episode, but presumptive evidence of pre-existing hyperthyroidism - unexplained weight loss which was then presumed to be associated with pericarditis itself - was present at the first episode of pericarditis. The diagnosis of Graves' disease could be made on the second episode of pericarditis due to clinical suspicion of hyperthyroidism. There have been no previous reports of recurrent pericarditis associated with Graves' disease. However, according to the pathologic background of the disease, recurrent pericarditis may well be a complication of Graves' disease as in our case.

No tissue diagnosis of the pericarditis revealing lymphocytic in-

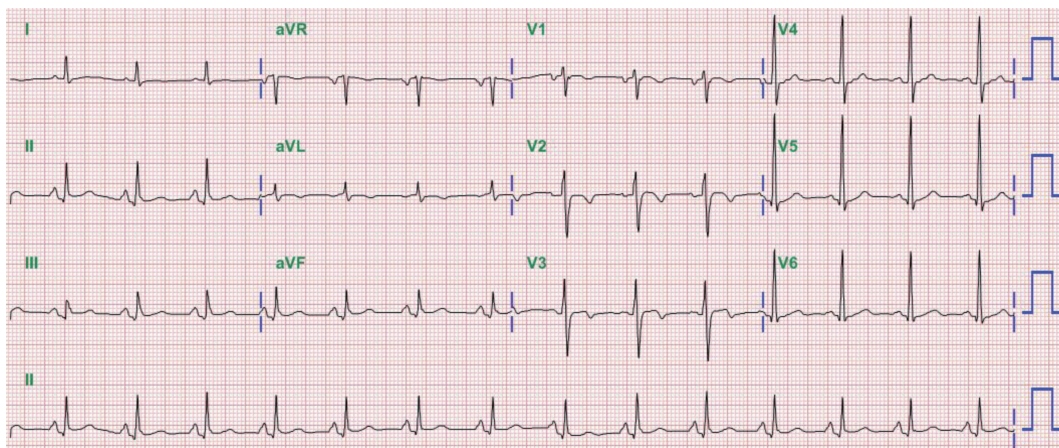


Fig. 4. Electrocardiogram at 3 months of follow up showed normalization of ST-segment elevation.

filtration was made in our case, but the course of the patient who did not develop any more recurrence with management of the thyroid problem accounts for a high possibility of the relationship between pericardial inflammation and Graves' disease. Our report may serve as evidence to guide the clarification of the underlying etiology in many cases of pericarditis where etiologic diagnosis cannot be made, and aid in management of recurrent pericarditis. Nevertheless, the relationship between recurrent pericarditis and Graves' disease may need more large-scale studies for elucidation of the association.

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